Venlafaxine-Induced Severe Sleep Bruxism in a Patient with Generalized Anxiety Disorder

Sir,

Sleep bruxism is a stereotyped movement disorder, which occurs during sleep characterized by teeth grinding.[1] The imbalance of neurotransmitters, including serotonin, norepinephrine, and dopamine during the nonrapid eye movement (NREM) phase of sleep is a postulated mechanism of nocturnal bruxism and the medications affecting these neurotransmitter systems can potentially induce this NREM sleep disorder.[2] Here, we present the case of a patient with generalized anxiety disorder who developed severe sleep bruxism with a serotonin-norepinephrine reuptake inhibitor-venlafaxine, which resolved on substituting it with a selective serotonin reuptake inhibitors-escitalopram.

A 35-year-old businessman presented with 9 years history of persistent anxiety symptoms, generalized apprehension related to day-to-day events and constant sense of restlessness. He had significant autonomic symptoms such as palpitations and sweating of palms. He would report of difficulty to relax and this had caused significant dysfunction in his personal and occupational facets of his life. He was treated in the past with varying doses of benzodiazepines — clonazepam (0.5-1 mg) and alprazolam (0.25-1 mg). However, he would not take these medications due to sedation, which affected his work. He had undergone therapy with an emphasis on relaxation training. However, due to his busy schedule at work, he was unable to regularly attend therapy sessions. He was initiated on tablet venlafaxine with dose escalated from 37.5 to 150 mg/per day (dose escalation was 37.5 mg once in every 4 days). At 1 week after the initiation of venlafaxine, his wife reported that he started having very severe grinding of

teeth during sleep at night. When this persisted for a week consistently, he came back and mentioned that it would be unable to continue the medication with this concern happening. The medication was stopped and was substituted with escitalopram with its dose starting at 5 mg/day hiked up by 5 mg every 4 days to 20 mg/day given at night. Within 2 days of stopping venlafaxine, bruxism disappeared, and he did not have similar side-effect on escitalopram. Within 2 months of initiation of escitalopram, he reported of significant improvement in anxiety symptoms.

A potential hypothesis that explains the mechanism of bruxism is based on the role of cerebral neurotransmitter control over the sleep-wake cycle.[3] Even though, dopamine is the most implicated neurotransmitter,[4] the role of other monoamines is also highlighted as a mechanism, as evidenced by the use of the adrenergic system modulating agents such as clonidine and propranolol to alleviate bruxism.[5-7] Overall, brainstem release phenomenon from higher cortical control has been considered to be a compelling mechanism.[1] In our patient, bruxism occurred with a dual acting agent venlafaxine, which has effects on both the serotonin and norepinephrine neurotransmitters. The Naranjo *et al*. probability scale suggested a possible relationship between venlafaxine and this adverse event.[8] Indeed, venlafaxine induced nocturnal bruxism has been reported previously.[9-11] This adverse effect had a temporal relationship with initiating venlafaxine and it remitted on stopping this drug in our patient. It did not reoccur on substituting it with escitalopram, which has a very selective action on serotonin. This calls for an increased awareness among the practitioners, of this distressing adverse effect with venlafaxine.

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